INDUSTRIAL CLINICS

PHYSICIANS AND SURGEONS

1313 N.W. 19TH AVENUE • PORTLAND 9, OREGON

December 8, 1958

A I R M A I L

Robert A. Kehoe, M. D.
Director, The Kettering Laboratory
College of Medicine--Eden Avenue
Cincinnati 19, Ohio

Dear Bob:

Enclosed is Thermo-Fax copy of letter which I directed to you on June 22, 1954, and copy of your reply. I am writing again about the same patient because of interesting developments in intervening four years. Subject patient is a white male named [redacted], now 56 years of age and in a very sad state of disrepair. As related in my letter of June, 1954, Mr. [redacted] was treated in hospital with split graft to the right shin and this graft healed rapidly and well. His fully demonstrated and rather profound lead intoxication apparently caused no interference with healing. No adverse effect followed blood transfusions. I followed him as an out-patient for approximately six months and health improved steadily while away from work as a burner. Continued personal stress arose from inability to find other work and he developed a chronic state of discouragement; late in November, 1954, he developed an acute duodenal ulcer requiring hospitalization. During the six months' period from May to November, he had continued to excrete urinary lead at level of 250 to 350 micrograms per liter. By late November at time of onset of acute duodenal ulcer he had for the first time reached level of 90 micrograms per liter of urine.

Treatment at hospital for ulcer resulted in rapid relief of discomfort. When I reported presence of ulcer to State Industrial Accident Commission, the Commission hastily advised that it had no responsibility for his ulcer. It was my honest opinion that this was a stress ulcer directly related to problems attaching to untoward combination of lead poisoning, burn and persistent unemployment. No allegation was made that ulcer had connection with lead poisoning per se. In absence of any financially responsible agency, the patient immediately discharged himself from hospital and I lost track of him for thirteen months.

In January, 1956, Mr. [redacted] came to this office having fallen at work at a local shipyard. Injuries included fracture of one wrist and the opposite shoulder and we put him in the hospital for this problem. At the time he again had complaints of malaise, backache and leg ache. Questioning revealed he had again engaged in burning work in preceding two to three months. New urine samples were sent to Oregon State Board of Health Laboratory for lead determination and analysis showed urinary levels of 240 and 175 micrograms respectively with one plus and two plus porphyrin tests on respective samples. These samples were taken two weeks and

N 6267

four weeks after occurrence of fracture of wrist and shoulder. Because of these elevations of urinary lead excretion I decided to experiment with oral calcium EDTA in amount of one gram three times each day. Patient's cooperation was quite uncertain during the next several days but he did achieve immediate elevation of porphyrins over a period of three weeks and also ran daily urinary lead excretion levels of 520, 600, 625 micrograms per liter and over a period of two weeks continued to excrete at levels of 260 to 500 micrograms per liter, returning within one month to 100 micrograms per liter. Urinary excretion remained at this level for several weeks further. By this time fractures were well healed and the patient had been discharged from care so far as traumatic injuries were concerned.

I saw Mr. [redacted] at three to six month intervals thereafter and on November 11, 1957, we were asked to examine him fairly completely. There was an interval history of acute ulcer perforation in October, 1956, necessitating emergency laparotomy and this was followed in April, 1957, by gastric resection at Veterans Hospital. In November, 1957, I again gave him a trial of oral calcium EDTA for three days. Urine samples before oral Versenate showed excretion level of 200 micrograms per liter and following day the level was 480 micrograms and the day after that, 320 micrograms. At all occasions since November, 1954, anemia had not been prominent and basophilic stippling not present in any degree. Hemoglobin has hovered between 90 and 95 percent and around thirteen grams.

Mr. [redacted] has again been examined fully and this time in hospital for three days in September, 1958. In the interim, he has fallen at home and suffered fracture of one hip and of lumbar vertebra IV and these have received treatment at University of Oregon Medical School County Hospital. In my most recent study of September, 1958, at the hospital, blood counts have been within normal limits. Urine samples have been collected on 24 hour basis; for one day before starting a trial with calcium EDTA sample showed 130 micrograms per liter. The following day I administered one gram calcium EDTA intravenously and the 24 hour sample immediately following showed 1,640 micrograms per liter. A further one gram intravenous dose of calcium EDTA was given the next day and the ensuing 24 hours produced lead urine excretion at the rate of 660 micrograms per liter. Versenate was then discontinued and the following day lead excretion returned to 140 micrograms per liter and on successive days it was then 220 micrograms and 150 micrograms. Five days later the level was 170 micrograms and at weekly intervals thereafter the excretion was 120 and 90 micrograms respectively. Because of confusing variations in laboratory results in this and other cases in past, I have not tried to follow blood lead levels.

I apologize for great length of this summary. I have a dossier nearly two inches thick on Mr. [redacted] and it seems obvious that it will continue to grow. Mr. [redacted] also has considerable generalized atherosclerosis, slight blood pressure elevation commensurate with age and a little albumin of urine but no evidence

of kidney retention. My questions at this time relate to some unresolved problems.

First, Mr. [redacted] has shown a rapidly deteriorating health picture and, at all times, mental deterioration many years in advance of his chronological age. As I have contemplated a number of other men working in similar recurrent exposures who have shown various degrees of lead intoxication through eight or ten years, I think I discern a common denominator of mental dullness and poor attention to detail and faulty memory which raises suspicion that chronic lead exposure causes chronic cerebral depression. I have no long-time way of evaluating this and it is purely a clinical impression. I do not know what mental levels may have been in the same individuals and I am sure they have never been measured. This particular aspect of chronic lead exposure and its potential for the cerebrum offers possiblility for a long-term study in some controlled employee group. Does the observation concerning lowered mental function jibe with your experience?

Secondly, do you have information relating to normal lead excretion rates under calcium Versenate stimulation in those presumably without unusual recent lead exposure? I have figures which were presented originally by Dr. Harriet Hardy in her article in J.A.M.A. of April 3, 1954.

Thirdly, can you refer me to some reliable source which discusses normal urinary lead excretions followed over a considerable period of time in a relatively large series of lead exposed workers? Is there experience which reveals approximate longevity of unusual lead excretion in those who may have been removed from exposure? I shall appreciate any comment you care to offer.

My wife and I have appreciated the opportunity to get to know you and Mrs. Kehoe, your reaction to our efforts and your lovely messages since return home. We hope your next Portland visit will not be long delayed. Solid Cincinnati is, of course, on our itinerary when and if we get East--of Chicago.

Your friend,

Forrest E. Rieke, M. D.

FER:fb

Enclosures (2)

DEPARTMENT OF ... AND ...

August 26, 1951

Dr. Forrest ... ieke
... Service for Industry
... Northwest ... Medical Street
... 10, Oregon

Dear Doctor ...ieke:

... several ... from the laboratory, one of which ... had ... ...cure ... ...atedly. I trust that I shall not b... lacking in courtesy, on account of my delay in rep... of June 22nd.

... will be of little use to you... o both of your questions is ... rs, but I've had no experience ... which bears pragmatically on e...

... e blood transfusion in the therapy of lead ... that I see no reason why such therapy should ... re good reasons for doing so. In one case of lead ... uodenal ulcer (rather a diagnostic problem) ... ed for the therapy of hemorrhage in the feeling ... and that there was no contraindication.

... there being difficulties, if the trans...

which might be found to exist among persons suffering from lead poisoning. Some of the latter have seemingly localized functional disturbances of short duration, while others (not very frequently in these days) appear to be suffering from some very generalized dysfunction. It might be supposed that conditions of the latter type would be incompatible with satisfactory healing, but again I should wish to see some rather concrete evidence, rather than to indulge in theorizing.

Very truly yours,